DOCUMENT RESUME

ED 118 382 SE 020 179

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TITLE Biophysical Basis of Electrocardiography.
PUB DATE Jun 75
NOTE 17p.; Paper presented at the Annual Meeting of the American Society for Engineering Education (Colorado State University, Ft. Collins, Colorado, June 16-19, 1975)

EDRS PRICE MF-$0.83 HC-$1.67 Plus Postage
DESCRIPTORS Biomedical Equipment; *Biophysics; Cardiovascular System; *Engineering; Engineering Education; *Higher Education; *Instruction; Instructional Materials; *Medicine
IDENTIFIERS *Electrocardiography

ABSTRACT

The generation of the electrocardiogram from a bioengineering standpoint is described, and this information is used to discuss its application to clinical instruction on electrocardiography. (MLH)

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AMERICAN SOCIETY FOR ENGINEERING EDUCATION

ANNUAL CONFERENCE, JUNE 16 - 19, 1975

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BIOPHYSICAL BASIS OF ELECTROCARDIOGRAPHY

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Introduction

The purpose of the paper is two-fold. In the first part we describe the generation of the electrocardiogram from a biophysical (bioengineering) standpoint. In the second part we utilize this information to discuss its application to clinical instruction on electrocardiography. An important objective is to illustrate the educational contribution of biomedical engineering to the life sciences -- with regard to a basically "engineering" process.

Electrocardiographic Biophysics

The basic structure of the heart is that of a stratified spiral-like configuration of muscle fibers circumscribing the two ventricular cavities. On a microscopic level the individual cells are arranged in a bricklike matrix. When a cell is activated it contributes a current density field throughout the entire body. That is, the body behaves as a passive volume conductor with the cell a source of emf. The electrical events give rise to contraction of the muscle so that the former may be diagnostic of pumping capability; hence, the interest in the electrical activity of the heart.

Since electrical events in nerve and muscle involve frequencies under 10,000 Hz the response of body tissue to endogenous currents can be examined through the application of a.c. signals over the range 0 - 10,000 Hz. Experimental results show it to be linear and resistive. Furthermore, for usual dimensions and this limiting frequency, propagation effects can be ignored. Consequently quasi-static formulations apply. Two important conclusions are (1) that at any instant of time the current field resulting

from many active cardiac cells can be found by superposition, and (2) that the current field of a single cell can be found as a solution to Laplace's equation, subject to the boundary conditions imposed by the cell and its environment.

With the latter conclusion in mind, consider the single cell, illustrated in Figure 1. We assume this cell to be lying in a uniform volume conductor

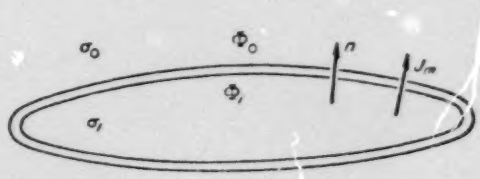


FIG. 1. The single cell in a volume conductor. The positive surface normal and positive transmembrane current are outward. The transmembrane potential V_m equals the intracellular surface potential relative to the extracellular surface potential; that is $V_m = \Phi_i - \Phi_o$.

of infinite extent of conductivity σ_o and whose cytoplasmic conductivity is σ_i and with the potentials Φ_o just outside and Φ_i just inside the bounding membrane. Now a characteristic of an active cell is that $\Phi_i - \Phi_o = V_m$ (the transmembrane potential) must be non-zero and, in fact, a function of position on the cell surface. Since biological membranes are very thin they may be considered as mathematical surfaces so that regarding the potential field Φ we must have

$$\nabla^2 \Phi = 0 \quad (\Phi \text{ satisfies Laplace's equation everywhere.}) \qquad (1)$$

$$\Phi_i - \Phi_o \neq V_m = 0 \quad (\text{Over the membrane surface S, } \Phi \text{ is discontinuous.}) \qquad (2)$$

$$\sigma_i \frac{\partial \Phi_i}{\partial n} = \sigma_o \frac{\partial \Phi_o}{\partial n} \quad (\text{continuity of current across the membrane}) \qquad (3)$$

$$J = \sigma E = -\sigma \nabla \Phi \quad (\text{Ohms Law in differential form})$$

In the above, n is the outward normal to the cell surface. Now define $\Psi = \sigma\Phi$. Then

$$\nabla^2 \Psi = 0 \quad \text{(everywhere)} \tag{4}$$

$$\Psi_i - \Psi_o \neq 0 \quad \text{(on S)} \tag{5}$$

$$\frac{\partial \Psi_i}{\partial n} = \frac{\partial \Psi_o}{\partial n} \quad \text{(on S)} \tag{6}$$

From potential theory the function Ψ satisfying the above conditions (solution to Laplace's equation, continuous derivative everywhere, discontinuity over the surface S) must be related to the discontinuity over S, namely,

$$\Psi = \frac{1}{4\pi} \int_S (\Psi_o - \Psi_i) \frac{\partial (1/r)}{\partial n} \, dS \tag{7}$$

where r is the distance from the source point to the field point, Solving for Φ gives

$$\Phi = \frac{1}{4\pi\sigma} \int_S (\sigma_o \Phi_o - \sigma_i \Phi_i) \frac{\partial (1/r)}{\partial n} \, dS \tag{8}$$

and σ takes the value σ_i for internal and σ_o for external fields. Equation (8) permits, in principle, the determination of the electric field (hence the current field) from knowledge of the cellular conditions. The source, in engineering terms, is described as a double layer (dipole moment per unit area) function lying in the membrane, oriented along the outward normal, and of strength $(\sigma_o \Phi_o - \sigma_i \Phi_i)$.

A determination of the double layer source for a cardiac cell from the biophysics of cell membrane processes is not possible with the current knowledge

of the field. In fact, spatial measurements of the electrical potential over a cell's surface have never been made. However, macroscopic measurements of fields within the heart muscle suggest that the sum effect of such double layers of many cells is itself a uniform electromotive (double-layer) surface which propagates uniformly from the inner to outer wall of the heart (roughly). If one imagines that excitation can freely pass from one cell to those adjoining it, as if there were intercellular contacts that permitted unimpeded flow of current between cells, then the double layer source of individual cells could combine to produce such an electromotive surface. In fact, utilizing a freely interacting model, one predicts the resultant electromotive surface to have a finite thickness and for it to consist of a dipole moment density that varies as an error function, and this is consistent with the macroscopic measurements. Unfortunately, this model is not completely consistent with known histology. The basic process of spread of activation as a wavelike phenomenon is, at least, an experimental fact. The location of such surfaces at successive instants of time has been determined from recordings from multiple-electrode needles inserted into the beating heart. An experimental determination shown in cross-section for the human heart is illustrated in Figure 2.

From an engineering standpoint, one should be able to evaluate the current field in the torso at some instant of time, given the geometry of the activation surface(s) at that moment and the geometry and conductivity of all elements making up the volume conductor of the body. The effects of inhomogeneities can, in fact, be considered by applying equations (4) - (6) at each interface between regions of different conductivity -- in which case one can confirm that,

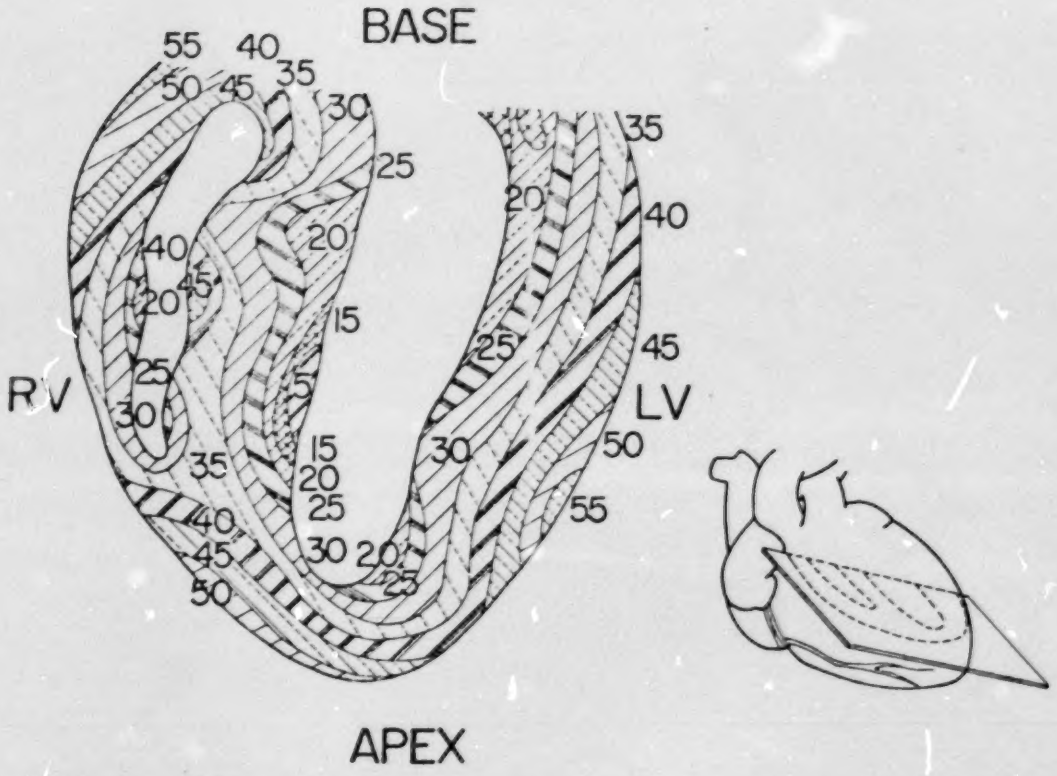


FIGURE 2 Isochronous lines of activation of the human heart. (From Durrer, D. et al., Total excitation of the human heart, *Circulation*, 41, 899, 1970).

due to surface S_j, a potential field

$$\Phi_j = \frac{1}{4\pi\sigma} \int_{S_j} \Phi_j (\sigma_j'' - \sigma_j') \frac{\partial(1/r)}{\partial n} dS \qquad (9)$$

results. In this equation σ_j'' is the conductivity on one side and σ_j' that on the other side of the interface while n is oriented from prime to double prime region. The effect of the discontinuity in conductivity is the introduction of secondary double layer sources of strength proportional to the potential times the discontinuity in conductivity. An important such

discontinuity is that at the torso surface, since this is an interface with the external (totally non-conducting) surroundings.

Although there are gaps in our knowledge, the aforementioned description of the generation of the electrocardiogram (the "forward problem") is reasonably well understood. While this matter is of great interest to the cardiac physiologist and of indirect interest to the cardiologist, it does not exactly address the problems of the latter. For the clinician, the electrocardiogram is the "given" information and it is the sources in the heart which must be determined therefrom (the "inverse problem"). Unfortunately, more than one source can (theoretically) give rise to the same electrocardiogram so that the inverse problem is ambiguous. And, for a given signal-to-noise ratio, the ambiguity is further increased. Much present effort is being directed to a study of this problem. If one is content to determine the net activity in a limited number of zones of the heart, and if one imposes from the outset some physiological constraints (e.g., activation surfaces are directed outward from the heart), then some limited success can be obtained. But more work is needed to sharpen our quantitative understanding of the capabilities and limitations of such multiple source inverse electrocardiography.

Simplifications and Approximations

The inverse problem can be simplified immeasurably if one is willing to make two assumptions. The first is that the torso is homogeneous. The second is to ignore the distributinn of dipole elements in the activation surfaces and consider the generator as the vector sum of all momentarily

existing double layers, that is, as a single dipole (whose orientation and magnitude is free to vary as a function of the progressing activity). In this case a simple relationship exists between the heart dipole source (call it $\bar{H}$) and the voltage, measured between two points (say a and b) on the body surface (V_{ab}). For suppose that $\bar{H} = 1\bar{a}_x$ (unit magnitude in the x direction) produced a voltage l_x in "leads" (a,b), and $\bar{H} = 1\bar{a}_y$ a voltage l_y, and $\bar{H} = 1\bar{a}_z$ a voltage l_z. Then, because the medium is linear, when $\bar{H} = h_x\bar{a}_x + h_y\bar{a}_y + h_z\bar{a}_z$,

$$V_{ab} = h_x l_x + h_y l_y + h_z l_z \tag{10}$$

Since this is in the form of a vector dot product one can write simply

$$V_{ab} = \bar{H} \cdot \bar{L} \tag{11}$$

where

$$\bar{L} = l_x\bar{a}_x + l_y\bar{a}_y + l_z\bar{a}_z \tag{12}$$

The vector $\bar{L}$ is known as the lead vector and it is a property of the lead (i.e., electrode pair a,b) location as well as the heart vector location -- it is a property of the geometry. One can measure lead vectors using an electrolytic tank the shape of the torso.

From three independent leads (three independent voltage measurements) the unknown, but desired, heart vector components (h_x, h_y, h_z) can be found. Special leads can be located (or constructed by linear combination) such that $\bar{L}_1 = \bar{a}_x$, $\bar{L}_2 = \bar{a}_y$, $\bar{L}_3 = \bar{a}_z$ and these constitute an orthogonal lead system -- the components of $\bar{H}$ are then obtained directly. Since the heart vector is, in fact, a distribution of dipole elements it is desirable that the lead

$\bar{L}_1 = \bar{a}_x$ be independent of position of the dipole source within the region of the heart. This criterion can be satisfied only approximately with practical leads. Several clinical systems of "vectorcardiographic" leads are in use today and they include the McFee-Parangao, Frank, SVEC III, cube, and tetrahedron.

Most clinical electrocardiograms are obtained using leads whose lead vectors have not been optimally chosen -- but have been adopted out of historical precedents. The lead formed from the left arm (left wrist is roughly at the same potential and electrodes are easier to attach at this point) relative to the right arm is V_I and the lead vector turns out (from torso tank studies) to be roughly along the anatomical direction from right (arm) to left (arm). This corresponds to the vectorcardiographic x direction. The "precordial lead" formed from an electrode in the fourth interspace just to the left of the sternum relative to a reference electrode derived from the mean potential of left and right arms and left leg (the Wilson central terminal) is V_2. It is roughly in the anterior-posterior direction which is the vectorcardiographic z axis. V_5, a precordial lead from the fifth interspace and anterior axillary line relative to the central terminal is another x lead. The left foot relative to the central terminal corresponds to a superior-inferior, or y-oriented, lead vector. In this way the scalar leads of conventional electrocardiography can be put into a more contemporary form as constituting an uncorrected (with regard to magnitude) but roughly orthogonal lead system permitting some quantitative statements about the heart vector. One major difficulty with vectorcardiographic use of the ECG is that clinical records are frequently taken sequentiallly, thus permitting only guesses regarding isochronous events.

The lead vectors corresponding to left and right arms (V_I), left leg and left arm (V_{III}), and left leg and right arm (V_{II}) were considered to form an equilateral triangle (Einthoven triangle). From torso tank experiments the triangle is seen to deviate considerably from this ideal. There is, of course, no reason to expect the lead vector to correspond to the vector joining lead locations -- in fact, the magnitude of the lead vector depends on the relative location of the source.

The above details are set in the larger electrocardiographic process. This begins with the automatic depolarization of cells in the heart's SA node until their threshold is reached. A consequence of their firing is the initiation of local currents which cause the activation of adjacent cells so that the process spreads throughout both atria. Special conducting cells (tracts) convey the activity to the AV node (the sole interconnecting link with the ventricles). Activity passes into the specialized conduction tissue of the common bundle and emerges, considerably delayed, in the ventricular septum -- thence to follow right and left bundles, and finally to reach the inner surface of the right and left free walls, as well as the septum, via the arborized Punkinje tissue. This initiates the activation of the ventricular walls and septum and gives rise to the activation wave already commented on. The temporal sequence of events is quite definite physiologically and the surface electrocardiogram reflects this. Thus, as illustrated in Figure 3, the P, QRS, and T waves are reliable features which characterize atrial and ventricular activity and ventricular recovery, respectively. Their time intervals and durations are relatively easy to

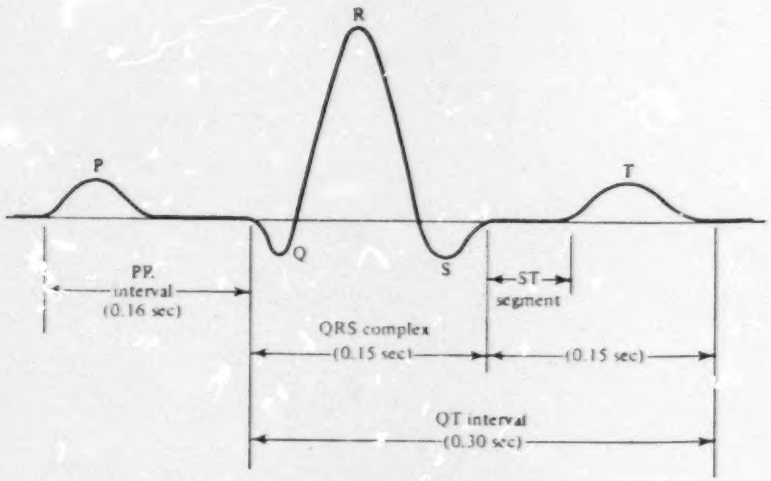


Fig. 3 Significant features of standard (scalar) electrocardiogram.

measure and, when outside the normal range, are amenable to straightforward explanations based on the normal pattern described above.

The Clinical Electrocardiogram

While it is not the purpose of this paper to consider clinical diagnosis of electrocardiograms we do wish to consider how the above material can be presented as useful background for such clinical studies. The description of the heart as a complex source is not useful since we do not yet have the tools to evaluate it in inverse electrocardiography. However, the single heart vector (heart dipole) is realizable and this should (and does) form the basis for clinical treatments.

By heart vector we mean a single dipole with a certain orientation and strength. The nature of a dipole source in an unbounded region can be described by a graphical presentation of its current flow field, as shown in Figure 4. This is useful for a graphical appreciation of the dipole field.

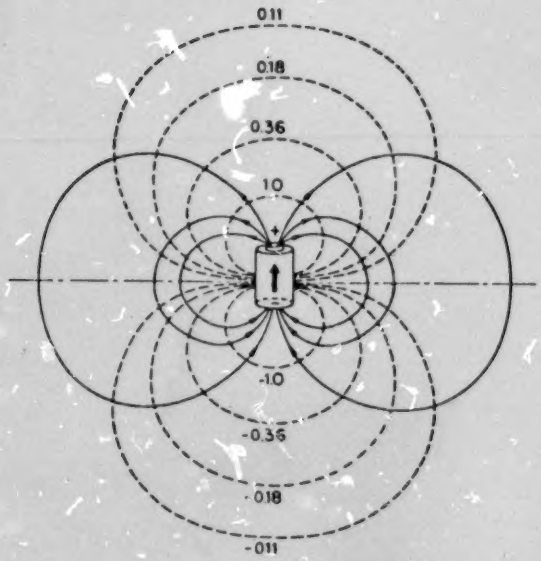


Fig. 4. A dry cell 'battery' is submerged in a conducting fluid. Current flows out of the positive terminal and returns to the negative terminal along the paths shown. The vector (heavy arrow) shows the orientation of the battery and, correspondingly, of the field. Under these conditions, since the battery provides a source-sink combination it is characterized as a dipole. The vector representing this (dipole) property of the battery has orientation (from negative to positive within the battery) and magnitude (strength of the dipole related to the battery emf). Several interrupted lines of equipotential, and their relative values, are shown.

This physical example is analogous to the electrocardiographic situation. In the latter case, the active muscle in the heart constitutes the dipole generator. Current flows away from the positive region (leaves the heart) into the torso volume conductor and returns to the negative region of the heart. Since the region of activity shifts from moment to moment, the dipole characterizing it is continually changing orientation and magnitude, the current flow field and associated surface potentials change concomitantly.

The notion of the lead vector is intuitive and serves as a basis for the interpretation of the voltage recorded by any single lead and its relationship to the heart vector. The locus of the heart vector is the vector loop which is, in fact, the goal of conventional inverse electrocardiography. As practice in construction of lead voltages from a known heart vector (or vice versa) such construction as shown in Figure 5 is useful. That the lead voltage is the dot product of heart vector and lead vector leads to discussion

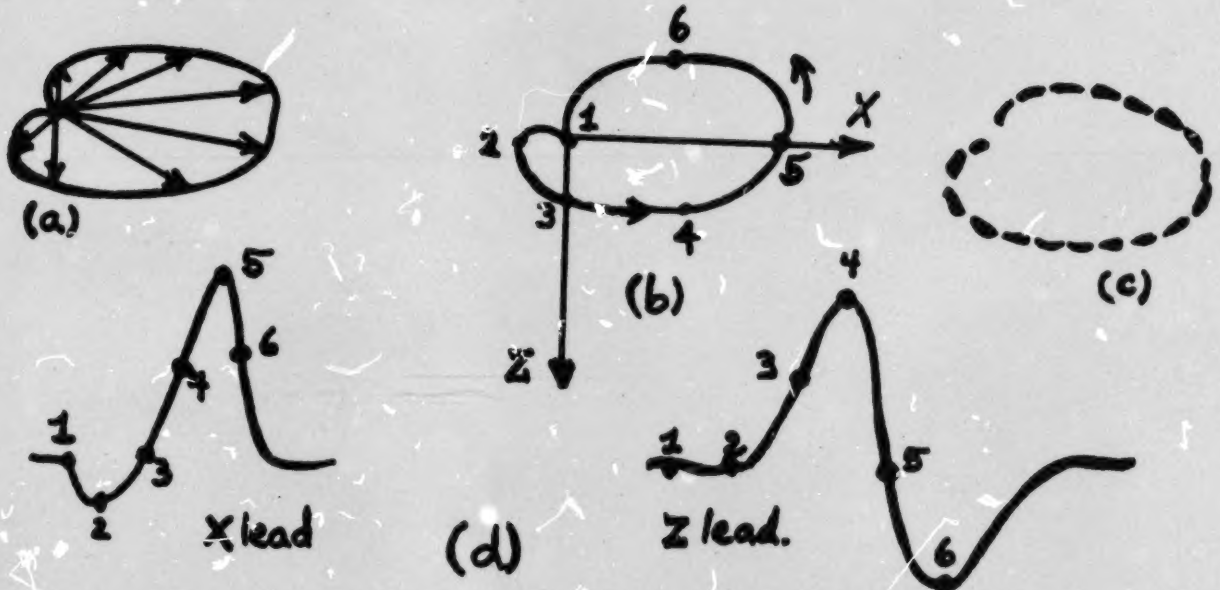


Fig. 5 (Legend) In (a) several successive heart vectors are depicted as well as the locus of the tip of the heart vector (b) the vector loop alone is shown, the arrows indicate the direction of inscription. The heart vector at the instant ta is drawn from the origin to the point corresponding to ta. (c) Electronically the vectorcardiogram can be blanked at regular intervals (normally .002 seconds apart) for timing purposes. In addition each segment is given a teardrop shape by electronic means inscription being revealed in this way (from pointed to blunt end). (d) The voltage that would be measured by an x or z directed lead is the projection of the loop (the heart vector, actually) on the lead vector OX or OZ as a function of time. Point 1 is the beginning (see (b)). At point (2) the heart vector is directed to the right, consequently the projection on x is maximally negative while there is zero projection on z. At point (3) the heart vector is in the anterior direction so that OZ is positive while OX is zero. At 4 the maximum positive projection on z is reached while the x voltage is still increasing. At 5 the x component is maximum positive (vector to the left) and no z voltage is attained. At (6) the x voltage is reduced while the z voltage is maximally negative.

of vector projection. Explanations for positive or negative going voltages can be given in terms of the necessary orientation of the heart vector.

At this point it is useful to review the normal activation sequence. Excitation begins on both septal surfaces, although a bit earlier in the left.

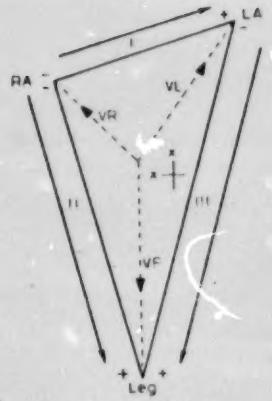
Thus, within the septum, activation (hence dipole surface orientation) from both right to left and left to right exists, but the predominance is left to right. (This means that the net heart vector should have a negative x component, initially.) The activity starts about two thirds of the distance down the septum, near the termination of the right and left septal bundles (consistent with the known anatomy). After septal excitation has begun, activity moves out to the inside of the free wall and to the apices (via the Purkinje system). On the right side this is mainly anterior, following the Purkinje spread, whereas on the left it is both anterior and posterior. The septal activity spreads slowly up the septum. The first large mass of ventricular tissue to be depolarized, following the septum, is the inferior, anterior leftward portion of the right ventricle. Although posterior spread in the left ventricle begins relatively early, most of the posterior left ventricle remains to be depolarized after apical depolarization is completed. After the apical myocardium has been excited much of the remainder of the excitation wave is outward. The lateral walls of the right ventricle are depolarized ahead of the thicker left ventricle. The final areas to be activated are the posterior left ventricle, the posterior-basal left ventricle and the superior septum.

From this electrophysiological picture the normal locus of heart vector can be roughly correlated with the underlying double layer sources. In this way vectorcardiographic interpretation need not be entirely empirical but can rest on a realistic physiological picture. The consideration of a net heart vector as the resultant of separate vector activity in the right

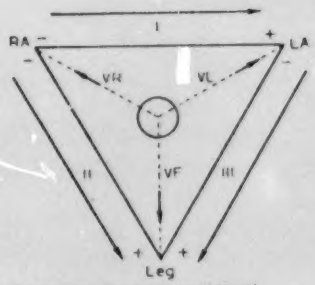
and left ventricles is an approximation since the vector regions are clearly separated and not at the same point. This is the basis of the approximate nature of the dipole representation of heart activity and is important to point out. (It explains the non-dipolarity of the heart, particularly noticeable in mid-QRS). The mechanical counterpart of a force couple applied to a rigid body yields a net zero resultant force; but, nevertheless, a torque exists. This has its counterpart when summing isolated activation vectors in the heart to produce a net heart vector, a process that ignores the higher order moments that can significantly contribute to the electrocardiogram.

The nature of lead vectors -- their approximate relationship to the physical location of electrodes -- can be explained. It can then be pointed out that the Einthoven triangle (see Figure 7) is, indeed, an idealization. The actual lead vectors arising from electolytic tank studies can be provided as a contrast -- as we show in Figure 6. In this way some further appreciation of care in the interpretation of the vector loop can be introduced.

Electrocardiography has proved its value as an empirical tool. Biophysical studies in electrocardiography have as their goal the development of increased diagnostic capabilities using sophisticated engineering techniques. Some of these developments and concepts are useful to the framework of clinical electrocardiography, as we have attempted to point out in this paper.



6 Burger triangle, scalene



7 Einthoven triangle, equilateral

Figure 6. The Burger triangle. The sides of this triangle are the lead vectors for V_I, V_{II}, V_{III}, as determined from an electrolytic tank measurement. The triangle, actually, is not in the frontal plane.

Figure 7. The Einthoven triangle. This is an idealized equilateral triangle composed of the lead vectors of the limb leads.

Acknowledgements

Thanks to Dr. Jerome Liebman, Professor of Pediatrics at Case Western Reserve University Medical School, with whom this writer developed syllabus material for an introductory lecture in electrocardiography for second year medical students. This paper was prepared with support from N.I.H. grant HL 10417 and N.S.F. grant GK 43464.